

Original Research Article

Avoidance of junk food during pregnancy, lactation and early years of child life:
A step towards the prevention of attention deficit hyperactivity disorder

Abstract:

Background: Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder which leads to a myriad of psychiatric and stoical consequences. **Aim:** Analyze the relationship between junk food and soft drinks (JFSDs) consumption of pregnant and lactating mothers and their children and the development of ADHD and related disorders among children. **Methods:** Primary school children in Kohat, Khyber Pukhtonkhuwa Province, Pakistan were surveyed. **Results:** Among 1200 children interviewed, 752 were entered into the analysis. The children's JFSDs intake pattern is as follows: 144 (19 %) mild, in 144 (19 %) moderate, 464 (61.70 %) excessive. The mothers' JFSDs consumption pattern was 181 (14.1%) mild, 209 (27.8%) moderate, 362 (48.1%) excessive. All levels of JFSDs consumption in children were significantly associated with variable degrees of ADHD or the related conditions [$\chi^2(df=3)17.5, p 0.0006$]. Also, all levels of JFSDs consumption of the mothers were significantly associated with JFSDs in respective children [$\chi^2(df=2), 13.948, p 0.0009$]. Boys were consistently at greater risk for ADHD and related psychological disorders [$\chi^2(df=3), 8.17, p 0.042$]. **Conclusions:** The frequency of ADHD and related psychological disorders among young children increase with increased JFSDs consumption of the children. Children to mothers used to consuming JFSDs during pregnancy or lactation tend to be JFSDs consumers. Cultural and economic factors could impact the likelihood of ADHD variably between both genders in Pakistani child populations.

Keywords: ADHD, students, junk food, Kohat, Pakistan.

Introduction:

Attention deficit hyperactivity disorder is a chronic, pervasive, childhood disease, characterized by a persistent pattern of inattentive behavior and/or hyperactivity-impulsivity that is more frequent and severe than is typically observed in individuals of the same developmental level [1]. Although there is no global consensus on the prevalence of ADHD in children, adolescents and/or adults, meta-regression analyses have estimated the worldwide prevalence at between 5.29% [2] and 7.1% in children and adolescents [3], and at 3.4% (range 1.2–7.3%) in adults [4]. The prevalence of ADHD in very young children (aged <6 years) or later in adult life (aged >44 years), is less well-studied [3]. Further, the prevalence rates of ADHD vary depending on several demographic, health status and clinical determinants. Whilst ADHD was once considered to be a childhood disease with a decline in symptoms during maturation to adulthood, it is now acknowledged to persist into adulthood in an estimated 50–66% of individuals [5]. Gender-wise, a higher prevalence of ADHD is often reported in males [3]. Children with inattentive behavior problems make careless mistakes with school work, find it hard to persist with tasks and are easily distracted. Those with problems in the area of hyperactivity/impulsivity often fidget and talk excessively, interrupt others, and are constantly restless. Based on the predominance of any of these morbid behavior patterns can verify ADHD into three different categories and hence

disease prevalence also varies. Clinically, patients with symptoms of both inattentiveness and hyperactivity-impulsivity are diagnosed with ADHD- combined type; those with primarily inattentive symptoms are diagnosed with ADHD- predominantly inattentive type; and those with primarily hyperactivity-impulsivity symptoms are diagnosed with ADHD- predominantly hyperactive-impulsivity type [6]. The combined ADHD type is considered most prevalent in children, adolescents and adults [3,7]. Further, ADHD is often present alongside comorbidities such as oppositional defiant disorder, conduct disorder (see later), anxiety disorder, personality disorders and depression which may further complicate understanding of true prevalence rate [8,9,10] s. Geographically, ADHD affects individuals across regions worldwide. The Diagnostic and Statistical Manual of Mental Disorders- 5th edition (DSM-V) suggests that cultural attitudes towards the interpretation of behavior may contribute to differences in prevalence estimates across studies [11]. Otherwise, the Centers for Disease Control and Prevention (CDC) more recently reported that nearly 1 in 10 children aged 4 to 17 years have parent-diagnosed ADHD (representing 5.4 million children in the US, half of whom are actively receiving medication) [12]. The disorder involves a group of behavioral, functional, emotional, and mental manifestations affecting the child's activity-quiescence equilibrium, attention span, focusing ability, wisdom and keenness to not err in social settings.

Disease Burden: The burden of ADHD overwhelm all environmental elements surrounding the affected child, including families, caregivers school, friends, schoolmates/teachers and colleagues, not to mention the often devastating health and social burden ADHD victims themselves have to tolerate. Not uncommonly, the impact of ADHD can devastate the child's life at home, school, and peer community. The annoying effect of ADHD upon the patient's sphere may be so disrupting to the point that it may put a strain on relationships with family members [13]. The behavioral symptoms associating ADHD, in terms of inattentive, hyperactive, and impulsiveness specifically lead to impairment in the parent - child relationship and contribute to increased stress among parents of children with the disorder [14]. Over time, parents may develop maladaptive and counterproductive parenting strategies to deal with these problems. [15]. On the run, children with ADHD experience persistent symptoms and functional impairments into adolescence; persistence can be predicted from psychological adversity and psychiatric comorbidity. At each stage of life, the impact of ADHD on functioning may vary. Should ADHD consequences continue through adulthood, the impact may well be felt in the workplace [16,17]. Eventually, the impairment associated with ADHD over the lifetime can have a significant impact on quality of life (QOL). Also ADHD can be associated with substantial financial burden for individuals, families and societal healthcare services.

The relationship between ADHD and dietary habits has received attention of researchers, worldwide [18]. In essence, diet alone is not the driving force behind the multiple behavioral and cognitive symptoms that plague children with ADHD. But studies have renewed interest in whether certain foods and additives might affect particular symptoms in a subset of children with ADHD [19]. Traditional research finds no support for radical diets like the Feingold diet, which eliminates nearly all processed foods as well as many fruits and vegetables, for the majority of children with ADHD. And there is no easy way to identify the few children who might benefit from diets that prohibit particular foods [20]. Yet parents, and some researchers, wonder if more modest dietary changes could supplement standard multimodal treatment that includes behavioral therapy and other evidence-based psychotherapies, school support, medication, and

parent education. The influence of diet upon the development of ADHD symptoms has been tracked as early as the fetal phase of children development. Epidemiological studies have shown that infants exposed to an increased supply of nutrients before birth are at increased risk of becoming overweight or obese in later life [21,22]. Further, the effect of junk food consumption by mothers-to-be ewes upon the eating attitude of respective newborns lambs during early life and as they grow up has been recently asserted. Experimental research led by Beverly Mühlhäusler in Australia is the first to show such effect at this early age of a child's life [23]. Inferences from Mühlhäusler et al., work indicate that junk food during pregnancy leads to “programming” of the ewes’ lambs-to-be and they become addicted to a high fat and high sugar diet by the time they are weaned [3]. In these experiments, it was found that a junk food diet in pregnancy and lactation desensitized the normal reward system fueled by these highly palatable foods. The offsprings in these studies were less sensitive to opioid hormones (“feel good” hormone); which are produced by the body as a “reward response” (in response to fat and sugar) indicate that the “reward pathway” (opioid signaling pathway) in the offspring was less sensitive than those whose mothers were eating a standard diet. Experimentally too, fetal exposure of maternal junk food in rats seriously interrupts the neuronal pathways of the mesolimbic reward center by altering the receptor expression in these specialized areas of brain and as a result the offsprings of these rats prefer fatty food intake over other foodstuffs [24].

Effects of junk food on neurological and behavioral health: The term junk food was explained by Michael Jacobson for certain categories of food which have either little or no nutritional value or have nutritional value but contain unhealthy ingredients: artificial colorings (AFCs) and preservatives; or completely unhealthy for use [25]. As far as the effect of junk food on the individual’s health, the provision of hyperactivity and or inattention disorders, specifically ADHD particularly in children has been a matter of extensive research in a variety of community and experimental settings. The burden of these disorders upon the community and the individuals warranted diligent work to mitigate these risks. For instance, ADHD alone affects up to 10% of children in the United States, and the prevalence of this disorder has increased steadily over the past decades [26]. The exact cause of ADHD is unknown. However studies suggest that it is associated with a disruption in dopamine-mediated signaling whereby dopamine D₂ receptors are reduced in reward-related brain regions [27]. This same pattern of reduced dopamine signaling is observed in various reward-deficiency syndromes associated with food and drug addiction, as well as in obesity.

Some researchers concluded that if mothers refrain themselves from eating junk food during pregnancy can prevent the abnormal neuronal circuitry in the mesolimbic reward centers of the offspring, and hence increased junk food preferences in childhood and later in life [44,48]. Junk foods, in turn, by large contain toxic ingredients, which are incriminated in the development of inattention/hyperactivity and ADHD symptoms. Many studies recently have been working in this track and conclude that food preservatives, particularly AFCs are causing increased ADHD symptoms among children in the general population [29]. However, there are still a controversy about such relationship and its strength [30-34]. The issue is that AFCs are mainly present in junk foods [35-39]. The effect of AFCs on brain function mimics that of addictive drugs, such as cocaine and heroin which interfere with neuronal activity of brain [40]. These pathways are seriously interrupted by the in-utero exposure of fetus to junk food, which increases the offspring’s preference for junk food [41] which will compel them to over-eat junk food later in life

as compared to others [42]. On the other hand, the role of many factors other than JFSDs in causing and influencing the tendency for ADHD has been addressed in a large body of research. Genetic factors have been suggested by many investigators as a principal culprit for ADHD [43]. On the other hand, environmental factors have also been highlighted as risk for genetically predisposed individuals to the development of ADHD behavioral changes [44,45]. Yet, as a whole the remarkable increase in the liability and frequency of hyperactive in subjects with sustained junk food use has been consistently emphasized [46,47]. The theory of sagvolden et al. [48], still offer an explanatory model of ADHD anatomical and functional disturbances as being caused predominantly by a hypo-dopaminergic mesolimbic (affecting the mesocortical and nigrostriatal) system, leading to abnormal reward and extinction processes. In fact, most ADHD mechanisms studies and trials data have been derived from observations on rat species, and sagvolden et al. [48], model has been perceived by some as an oversimplification of ADHD pathophysiology. This criticism reflects the subsequent limitation of the use of animal models to explain higher mental disorders in human [49].

Presentation and complications of ADHD (and related disorders): According to DSM-IV diagnostic criteria, ADHD is a neurodevelopmental disorder characterized by impairment in the executive functions of brain that includes inattention, hyperactivity and impulsivity [50]. In practice, too, inattention, hyperactivity (restlessness in adults), disruptive behavior, and impulsivity are common ADHD criteria [51,52]. With this symptomatology profile, it is often difficult to define ADHD and related disorders, as it is hard to draw a line at where normal levels of inattention, hyperactivity, and impulsivity end and significant levels requiring interventions begin. Thereby, ADHD is diagnosed before the age of six and out of these diagnosed cases 60 % shows ADHD symptoms in their adulthood [53]. According to the DSM-V, symptoms must be present for six months or more to a degree that is much greater than others of the same age, [54], and they must cause significant problems functioning in at least two settings (e.g., social, school/work, or home).

As far as presentation, ADHD individuals could be predominantly inattentive, predominantly hyperactive-impulsive, or a combination of both presentations. [52] Clinically, inattentive ADHD patients are easily distracted, miss details, frequently switch from one activity to another, become bored with a task after only a few minutes, have difficulty learning something new, poorly completing or turning in homework assignments, daydream, and struggle to follow instructions [55]. Likewise, hyperactive/impulsive ADHD patents tend to be fidget and squirm in their seats, talk nonstop, touching or playing with anything and everything in sight, have trouble sitting still during school, have difficulty doing quiet tasks or activities, blurt out inappropriate comments, act without regard for consequences, and interrupt conversations or others' activities [55]; On the other hand, ADHD associated disorders occur in about two-thirds of the time. Some commonly associated conditions include a) learning disabilities (20–30% of ADHD children) with developmental speech and language disorders [56], b) “Tourette syndrome” [55], which encompasses a broad spectrum of motor and vocal tics of variable severity and chronicity [57], c) “oppositional defiant disorder” (ODD) and conduct disorder which mainly involve antisocial behaviors such as stubbornness, aggression, temper tantrums, lying [58], and stealing, d) “restless legs syndrome”, which is often attributed to iron deficiency anemia associated with ADHD mostly in boys [58], and e) sleep disorders (either as ADHD comorbidity or as a side effect of ADHD medications). Further, ADHD is a predisposing factor of other psychiatric conditions such as antisocial personality disorder,

bipolar depression (BPD) symptoms [59], and substance abuse [60]. Other consequences include low educational level, poor socioeconomic conditions and tendency towards criminality [61,62].

Sex variation: Whereas junk food impacts the executive functions such as inattention, hyperactivity and impulsivity [50], observations also suggest that males are at greater risk of developing neuro-psychological ailments related to such exacerbated neurological functions, namely ADHD symptoms or related varieties. Most studies on the impact of palatable food intake on the mesolimbic reward system have been conducted in males [24]. In their controlled trial on the effect of maternal junk food feeding of rat dams on altering food choices and development of the mesolimbic reward pathway in the offspring, Ong, et al. (2011) found that chronic intake of a palatable cafeteria diet (CD) and subsequent abstinence influence fat mass, food intake and key gene expression of the mesolimbic reward system unequivocally differently in males and females [24]. While chronic CD intake increased fat mass in all CD rats but body weight and chow intake were reduced during the period of cafeteria diet abstinence, probably due to reduced tyrosine hydroxylase (TH)- mRNA in male CD and CD withdrawal rats, but increased in female counterparts, together with a reduced dopamine active transporter (DAT), D1 mRNA in CD and CD-W females, but increased in CD males, compared to Controls. Likewise, μ -opioid receptor expression was reduced in CD and CD-W males but not females]. The study concluded that investigating sex differences in the neurobiological response to palatable food intake in future researches should include both sexes. This work was based on the hypothesis that JFSDs intake in the perinatal period may lead to increased food preference towards junk food among offsprings, and who in turn become liable to developing ADHD or related symptoms.

Management of ADHD: Caring about ADHD in children is a multidisciplinary process that involves several aspects, such as school intervention and social skills training, in order to help affected children come over their symptoms. Medical intervention and healthcare providers' assistance may definitely be needed with certain disease severity; however nonmedical behavioral and social aids, especially involving parents in treating their ADHD children proved highly effective. With this respect, "behavioral parent training" (BPT) has been envisioned as one of the most effective ways to change poor parenting and therefore provide most favorable environment for treating ADHD [63]. The BPH technique has been influenced greatly by the work of early researchers, such as Patterson and Gullion (1968) [64] and Forehand and McMahon (1981) [65], who focused their research on noncompliant and aggressive children. On the basis of such pioneering work of BPT, parents are taught to identify and manipulate the consequences of child behavior, and monitor problematic behaviors, reward prosocial behavior through praise, positive attention, and tangible rewards, and decrease unwanted behavior through planned ignoring, time out, and other nonphysical discipline techniques. Since ADHD commonly co-occurs with other childhood disorders, particularly ODD, conduct disorders, learning disorders, and internalizing disorders such as anxiety, and since behavior modification is now a well-established part of effective treatments for a wide variety of childhood behavioral and mental disorder (e.g., ODD, conduct disorders, anxiety, depression, and autism, indeed, BPT is a leading part of empirically supported treatments for all of these disorders, including ADHD [66]. School-based intervention is an important adjunct to BPT for families of children with ADHD. The issue is that ADHD involves cross-situational impairment, and virtually every child with ADHD will experience problematic school behavior and functional impairment in their social relationships with adults or peers and in their academic performance. That is why school has a pivotal role to play in reversing ADHD symptoms both among affected children and those who

are at high risk for ADHD. Interventions applicable at school setting include “brief behavioral consultations with teachers” [67], comprehensive and sustained school-based consultation and intervention [68], or the placement of a child in a classroom that include intensive contingency management strategies [69]. Moreover, researchers building state-of-the-art prevention programs for children at-risk for developing ADHD have coupled BPT with school interventions in studies with head start children and as part of a sustained, multimodal prevention program for diagnosed ADHD elementary school children and other ADHD prone peers [70]. Likewise, “social skills training” is another dimension where ADHD children are tackled in order to handle the interpersonal difficulties which are one of the hallmark qualities of ADHD they suffer [66]. Children with high levels of hyperactivity, noncompliance, or aggression are rated more negatively by peers on sociometric measures and are more likely to be rejected by peers. Thus, peer relationships are an important target of comprehensive treatment for ADHD. The “behavioral social skills training” (BSST) model includes those interventions focused on reinforcing the use of appropriate social skills (e.g., communication, cooperation, participation, validation). Further, greater effects were found for combined BPT and BSST treatment, which demonstrated improvements across home, school, and peer domains. [66]. From the pharmacological intervention standpoint, it is estimated that 75% of children diagnosed with ADHD are medicated with stimulants [71]. Stimulant medication has been shown to have large, beneficial effects on a number of outcome measures, including ADHD symptoms. Many studies have compared BPT to stimulant medication and combined BPT and stimulant medication. For example, the “multimodal treatment of ADHD” (MTA) study included three intervention groups: a) behavioral treatment (BT); included BPT and school interventions); b) medication management (MedMgt); c) combined BT-MedMgt (Comb); and a community comparison control (CC) group, of which two-thirds were medicated [72]. Although medication in this study was effective in reducing ADHD symptoms, for the socially valid targets of treatment (i.e., areas of impaired functioning), only the Comb treatment resulted in improved social skills and improved parent–child relationships, including harsh and ineffective parenting. Importantly, when parents were asked to rate their satisfaction with treatment; they overwhelmingly endorsed the treatment conditions that included BPT. These results are not surprising. Parents of children with ADHD commonly report problems with morning and evening routines, and these are typically times when stimulant medication treatment is impractical because of side effects such as insomnia. Therefore, even if a child is medicated for the entire school day and early evening, there are clearly times parents need behavioral strategies to help them manage their child’s behavior. The results of the MTA study further suggest that for a child on medication, BPT is required to increase the chances that the child behaves within normative limits and improves in important functional domains. Perhaps then, medication for ADHD may be best thought of as another possible enhancement to BPT for children and families who do not respond to the standard treatment. Whereas ADHD is associated with a host of family problems, it is unlikely that stimulant medication for children is sufficient to treat the multiple mental health needs and pervasive impairment common in these families. Indeed, it has been found that late afternoon stimulant dosing for children with ADHD did not result in improvements in parent mood and functioning [66]. Clearly, behavior modification is a necessary component of comprehensive treatment for ADHD.

Methods:

This school based retrospective study was conducted between September 2015 and January 2016 on 1200 children, 4 – 7 years of age, randomly selected from the primary school system of Kohat educational district, Khyber Pukhtunkhuwa, Pakistan. Twelve schools were included in the survey, six of them were private institutions and six governments' and all were randomly selected considering rural, urban, and gender wide distributions. Data collection took place at two study settings, school and home. Further, predesigned instruments and clinical interview of selected children would be utilized to collect study data. The instrument part consists of two sets of questionnaire forms. One set is to be distributed among school teachers and consists of a consent form and a self-reporting questionnaire reflecting DSM-IV diagnostic criteria for rating of children inattention and hyperactivity during school time. Another set consists of a consent form and three other predesigned questionnaires to be sent to the parents of the sample students (through the cooperation of school administration). The parent-administered questionnaires were meant to collect data from parents, as will follow: a) the first questionnaire sent to the mothers and addresses demographic criteria and different types of food intake, both healthy and junk during the course of respective pregnancy, (including first, second and third trimester; b) the second questionnaire covers food preferences of respective children so as to ascertain the child's healthy and junk food and their weekly food menu; c) the third questionnaire relates to symptoms of ADHD and related disorders, as derived from DSM-IV diagnostic criteria. The students' JFSDs eating habit for each sample unit could be divided into three levels: mild JFSDs eaters who use 1-20 junk food items per week, moderate JFSDs eaters who eat 20-30 junk food items per week, and excessive JFSDs eaters who eat 30 and above of junk food items. All questionnaires were designed and modified in local Urdu language. The questionnaire distributed among schoolteachers also includes the DSM-IV diagnostic criteria for the diagnosis of ADHD (and consists of 18 items), for the teacher rating of children hyperactivity level. The study questionnaires were collected after one month. The food taken by the mothers during pregnancy and the food preferences of children would be compared to see the effect of in-utero- and via lactation -exposure to different foods on the children preferences. Similarly, the hyperactivity scores recorded during the two settings, i.e., school (by the teachers) and homes (by the parents), were compared for the diagnosis of ADHD.

In developing the questionnaire instruments, these researchers aimed to examine available studies which of similar coverage. Thorough literature review and incorporating correlates of potential association with the study outcomes of our concern helped integrate those items most useful to enable examining the correlates of the study's best interest. The proposed questionnaires were then reviewed by a panel of community research experts and scholars in the same study field, e.g., including nutritionists, pediatric psychologists to assure maximum questionnaires' validity. Modifications of the questionnaires were done, based on the received experts' remarks. Extensive effort has also been done to ensure highest reliability levels possible. Therefore, pilot administrations were undertaken to measure the questionnaires' test-retest reliability levels. Fifty-six sets of questionnaire forms were distributed to teachers and parents to respond to (response-a) and reveal the food intake patterns and ADHD information of the respective children, as per the study design. (Thirty-one questionnaire sets sent to boys' school and 25 questionnaire sets sent to the girls' school in central Kohat educational district). The same questionnaires were given to the same pilot sample one week later (response-b). A panel of juries

was invited to judge the responses. Test-retest reliability assessments of the pilot questionnaires'' items were carried out using Spearman ρ . Obtained correlation coefficients for each pair of items (response-a; and response-b) were all significant on all correlations ($p<0.05$) and indicating strong correlation on most items (ranging between 0.77 – 0.90. The questionnaires take between 20-30 minutes to complete. Necessary clearance and formal approvals were obtained from the all concerned parties, including Kohat schooling administration, and Kohat school health department research ethics committee before the study. Onsite, teachers and students were informed about the aim of the study prior to the completion of questionnaire, indicating that participation was voluntary, as well as anonymity and confidentiality of the gathered data, and assuring that only generic outcome data might be disseminated through scientific channels.

Psychiatrist help was taken to confirm any hyperactivity symptoms of children, which would be labeled either as ADHD, inattention (attentive disorder), hyperactivity below threshold level (hyperactivity/impulsivity) and no sign of hyperactivity. In setting criteria for considering ADHD diagnoses using DSM-IV hyperactivity scoring, six inattentive symptoms and six symptoms of impulsivity and hyperactivity were consistently used. After confirmation of the hyperactivity level, it was compared with the food preferences of children to identify the relationship of hyperactivity with different types of food items. Thereby, three specific questions would be addressed to measure this relationship: "whether a hyperactivity diagnosis of a subject sample students is related to junk food intake", "if there any relationship of food preferences of the student is related to the food intake by the mother during pregnancy," and "whether avoiding junk food during pregnancy could be solution for the prevention of ADHD." Other than age and a school attendance record $\geq 75\%$, no boy or girl student would be excluded from the study. Also, in order to be included in the analysis, only returned questionnaires with 80% or more valid answers would be entered. Ultimately, each child surveyed was subject to five encounters, ADHD questionnaire the teacher in charge had to return, and three questionnaires for the mother to respond to, as above, and a clinical psychological interview. Therefore, in reporting the study results, we may often refer to any studied children as "study sample unit" or simply "sample unit".

Statistical analysis: Collected data and verified variables were all coded and entered into a Microsoft (MS) program with adequate backup. Descriptive statistics, e.g., numbers and proportions were displayed. Statistical analytical techniques were used, as appropriate. For the most research, the association between categorical variables, e.g., influence of maternal JFSDs intake during pregnancy or lactation upon on the level of respective children's JFSDs intake pattern could be assessed using the Chi square (χ^2) test of independence, (Fisher's exact alternate might not be used as no expected frequency of the tested subcategory cells was less than five), and so forth. (The JFSDs consumption encompassed three levels mild, moderate and excessive, as above). In the analysis, too, the study population sample may often be stratified by gender and geographical residence (as rural and urban) and in which case, the influence of the input of interest, e.g., JFSDs intake of the children upon the risk for ADHD or related disorders could be measured using χ^2 test technique. The "Statistical Package for Social Sciences" (SPSS) software for Microsoft- version-20 was used for the analysis. All tests were conducted at level of significance $\alpha=0.05$; results with p -values <0.05 will be considered statistically significant.

Results:

Out of 1200 children surveyed, 752 (62%) turned valid questionnaire responses. Boy students accounted 403(53.6%) and girls accounted 349(46.6%) were female students, yielding a male-to female ratio of 1.15:1.

Table 1: Distribution of the study children with behavior disorders by JFSD consuming pattern

Children's JFSDs consumption pattern	ADHD		Attentive disorder		Hyperactivity / impulsivity		No signs of ADHD		Total	%	Test statistic	p-value
	No.	%	No.	%	No.	%	No.	%				
Mild	5	0.6	21	2.8	24	3.2	94	12.5	144	19.1	$\chi^2(df=3)$ 13.50	0.0036
Moderate	9	1.2	18	2.4	19	2.5	98	13.0	144	19.1	$\chi^2(df=3)$ 17.50	0.0006
Excessive	33	4.4	128	17.0	87	11.6	216	28.7	464	61.7	$\chi^2(df=3)$ 18.34	0.0004
<i>Total</i>	<i>47</i>	<i>6.3</i>	<i>167</i>	<i>22.2</i>	<i>130</i>	<i>17.3</i>	<i>408</i>	<i>54.2</i>	<i>752</i>	<i>100.0</i>		

As in Table 1, among 752 children analyzed, their JFSDs consumption pattern was as follows: 144 (19 %) mild, in 144 (19 %) moderate, 464 (61.70 %) excessive. All levels of JFSDs consumption in children were significantly associated with a level of ADHD. For instance, 46 (6.1%) of moderate JFSDs consuming children had either ADHD, attentive disorder, or hyperactivity/impulsivity disorder [$\chi^2(df=3)$ 17.5, p 0.0006].

Table 2: Impact of mothers' junk food and soft drinks consumption pattern during pregnancy upon junk food and soft drinks consumption among their children

Mother's JFSDs consumption pattern during pregnancy	Children JFSDs consumption pattern						Total	%	Test statistic	p-value
	Excessive		Moderate		Mild					
	No.	%	No.	%	No.	%	Total	%		
Mild	133	17.7	24	3.2	24	3.2	181	14.1	χ^2 (df=2) 40.05	0.001
Moderate	122	16.2	43	5.7	44	5.9	209	27.8	χ^2 (df=2) 9.32	0.0095
Excessive	209	27.8	77	10.2	76	10.1	362	48.1	χ^2 (df=2) 13.95	0.009
Total	464	61.7	144	19.0	144	19.0	752	100.0		

Table 2 displays the mothers' JFSDs consumption pattern in the perinatal period, which was found to be mild in 181 (14.1%) mothers, moderate in 209 (27.8%) mothers, and excessive in as many as 362 (48.1%) mothers. All levels of JFSDs consumption of mothers were significantly associated with JFSDs in respective children. For instance, excessive JFSDs consuming mothers

produced 362 (48.1%) children with variables levels of JFSDs consumption [excessive in 209 (27.8%) children, moderate in 77 (10.2%) children, and mild in 76 (10.1%) children, [$\chi^2(df=2)$, 13.948, p 0.009].

Table 3: Distribution of the study children with behavioral disorders by residence and gender

Children's junk food and soft drinks consumption pattern	ADHD		Attentive disorder		Hyperactivity / impulsivity		No signs of ADHD		χ^2 (df=3)	p -value
By gender	No.	%	No.	%	No.	%	No.	%		
Male	27	3.6	85	11.3	72	9.6	219	29.1	8.17	0.042
Female	20	2.7	82	10.9	58	7.7	189	25.1		
By residence by gender										
<i>Urban</i>										
Total children	36	4.8	104	13.8	92	12.2	272	36.2	9.27	0.026
Male	19	3.8	53	10.5	49	9.7	161	31.9		
Females	17	3.5	51	10.1	43	8.5	111	22.0		
<i>Rural</i>										
Total children	11	1.4	63	8.4	38	5.1	136	18.1	8.56	0.035
Male	8	3.2	32	12.9	23	9.3	58	23.4		
Females	3	1.2	31	12.5	15	6.0	78	31.5		

Table 3 shows that male children were consistently at greater risk for ADAH and related psychological disorders, as such as ADHD 27(3.6%) v. 20(2.7%); attentive disorder 85(11.3) v. 82(10.9%), and hyperactivity /impulsivity 72(9.6%) v. 58(7.7%), [$\chi^2(df=3)$, 8.17, p 0.042]. All these figures add up to 184(24.7%) male children with variable degrees and types of ADHD, compared to 160(21.3%) female counterparts (Table 3).

Discussion:

This study was designed to be conducted on a sample of young students of Kohat population in Pakistan. A sample as large as 1200 students was first targeted to ensure maximum study representativeness of the target child population. This allowed adequate make up for the expected proportion of invalid data such as incomplete questionnaires responses and dropout. Early in the study, for instance, many parents refused to sign the consent form and opted to withdraw their children from the study cohort. Overall, 448 parents did not attend the survey, 392 out of which refused to take part in the study. These 392 were of the opinion that such studies are useless and wastage of time. A group of parents, which numbered 28 refused after reading and gaining knowledge about the study and thought they cannot disclose their personal information of food preferences, particularly during pregnancy and lactation. Out of the 448 parents 28 mothers could not recall their food preferences during the perinatal period so no data could be collected from them though they were willing to participate in survey and allow their children to take part in the study. Some of the parents numbered 94 were so enthusiastic that they added certain extra food items, which were not present in the list provided of the survey form. This was evidence that some of the parents were so interested to explore more and more about the behavior and activity level of their children.

During the study we found that mother's dietary habit mostly remained the same all through pregnancy, and also lactation and early period of respective child development. This consistent behavior of mothers, especially for JFSDs has significantly shown a strong association with their children food preferences in later life. The next ring in this JFSDs- AFCs chain is the probability of hyperactivity of children and then the predicted complications in terms of morbid behavioral health early in life [50], as well as the myriad of psychiatric disorders that may end up with substance abuse, BPD, and antisocial personality behavior [73]. In this study three JFSD-groups were identified, mild, moderate and excessive JFSD users. The junk food taken by the children is almost the same as the mothers' junk food eating behavior, including items such as chips, pizza, ice creams, candies, biscuits, chocolates, juices, pastries, burgers, chowmain, soft drinks, different fruit jellies, and the likes. Alarming, the majority (62.70%) of the studied children are excessive JFSD users, probably reason why some number of the sample frame parents refused to participate because they would have to disclose information about the mother's feeding attitude during pregnancy. Alarming, too, male students dominate the female students in mild JFSDs preference, and dominate more heavily moderate and excessive JFSDs consumption (2.2 times than female). The same gender pattern of junk food consumption had been reported in other studies. While some of these studies are criticized as they were only conducted on male recruits [24]. The gender variation status in Pakistan may be due to different reasons. Cultural and economic factors intervene. In rural Pakistan, men have more access to life opportunities, including entertainment and junk foods and soft drinks than women who are still more submissive in such a man dominant society. Kohat is no departure from rural Pakistan. Boys there have more freedom to move, hang around, and are more fortunate; having exposure to life opportunities, even if unhealthy such as getting JFSDs for leisure. Girls, in contrast, are way less fortunate; many may be more toward being home bound. Likewise, urban residents show greater tendency for JFSDs intake compared to rural nationals, probably due to the difference in the purchasing ability in favor of urban society. In the former, lower income, less hoteling trend, and more home-cooking, all may be leading causes.

In this study, the more the junk food used by the child the more they are prone to ADHD or ADHD symptoms are noticed in the child [23,74]. All together out of 752 there were 403 male students responded to the surveys as compared to 349 female students, 47 samples students were found having complete ADHD thus frequency of our studied population was 6.3 % which is almost similar to the 3-10% recorded by the CDC USA, which shows that ADHD has a strong correlation with eating habits of the children rather than culture and geography [75]. One more important strong positive relationship may be seen in mothers eating habits during pregnancy, lactation and six months after stopping lactation with that of their respective studied children. Mothers utilizing excessive junk food and soft drinks during those three stages had 27.8% of total children with excessive junk food and soft drink preferences and 10.2% as moderate. Such finding shows that eating habit of mother does have effect on the eating habit of the children. This association may well be supplemented by several additional factors identified in the study ranging from rural and urban distribution, education background of the mother, cooking habits of the mothers and eating outside habits. The male / female difference in JFSDs utilization pattern observed in this work which supports the cultural male dominance in Pakistani rural society makes perceiving AFCs as single source of ADHD less likely. Multiple other factors, e.g., family and mother eating habit, culture, access to junk food and socio-economic status, all play a pivotal role in labeling a child as ADHD. There were two main limitations of the study first being

cultural and religious taboo as questions were related to pregnancy and lactations, which was the main cause of less participation of mothers and students. Second study limitation was due to its retrospective nature therefore appropriate amount of junk food intake by mothers during pregnancy lactation and early years of child life cannot be calculated therefore frequency of intake by mothers according to weekly food menu was taken as one of the variable. Keeping these constraints in mind a systematic and detail prospective study might be needed. The most commonly used coloring agents in all these junk food studied were E122 (carmoisine), E129 (alura Red), E133 (brilliant blue) and E150 but the important limitation of the study was the failure of finding out which one AFC is causing hyperactivity and to which extent for which more research studies should be done.

In conclusion, avoiding junk food during pregnancy, lactation and early years of child development can decrease the child preferences for junk food which decrease the frequency of ADHD/hyperactivity. Nonetheless, prospective studies should be undertaken to identify the contribution of in-utero exposure, exposure during breast milk and provision during early years of child development to junk food preferences later in life. There is also a need to specify the specific AFCs and their doses, which are solely responsible for the development of ADHD/hyperactivity

References:

- 1 American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision. Washington, DC: American Psychiatric Association, 2004.
- 2 Polanczyk G, de Lima MS, Horta BL, Biederman J, Rohde LA. The worldwide prevalence of ADHD: a systematic review and metaregression analysis. *Am J Psychiatry* 2007; 164: 942-948.
- 3 Willcutt EG. The prevalence of DSM-IV attention-deficit/hyperactivity disorder: a meta-analytic review. *Neurotherapeutics* 2012; 9: 490-499.
- 4 Fayyad J, de Graaf R, Kessler R, Alonso J, Angermeyer M, Demyttenaere K, et al. Cross-national prevalence and correlates of adult attention-deficit hyperactivity disorder. *Br J Psychiatry* 2007; 190: 402-409.
- 5 Barkley RA, Fischer M, Smallish L, Fletcher K. The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. *J Abnorm Psychol* 2002; 111: 279-289.
- 6 Department of Health. Mental Health of Young People in Australia. 5 Depressive disorder, conduct disorder and attention-deficit/hyperactivity disorder. <http://www.health.gov.au/internet/publications/publishing.nsf/Content/mental-pubs-m-young-toc~mental-pubs-m-young-5>
- 7 Faraone SV, Biederman J, Weber W, Russell RL. Psychiatric, neuropsychological, and psychosocial features of DSM-IV subtypes of attention-deficit/hyperactivity disorder: results from a clinically referred sample. *J Am Acad Child Adolesc Psychiatry* 1998; 37: 185-193.
- 8 Nøvik TS, Hervás A, Ralston SJ, Dalsgaard S, Rodrigues Pereira R, et al. Influence of gender on attention-deficit/hyperactivity disorder in Europe-ADORE. *Eur Child Adolesc Psychiatry* 2006; 15 Suppl 1: I15-I24.
- 9 Kessler RC, Adler L, Barkley R, Biederman J, Conners CK, Demler O, et al. The prevalence and correlates of adult ADHD in the United States: results from the National Comorbidity Survey Replication. *Am J Psychiatry* 2006; 163: 716-723.

518 10 Friedrichs B, Igl W, Larsson H, Larson JO. Coexisting psychiatric problems and stressful life
 519 events in adults with symptoms of ADHD--a large Swedish population-based study of twins. *J*
 520 *Atten Disord* 2012; 16: 13-22.
 521 11 American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders,
 522 Fifth Edition. 2013.
 523 12 Centers for Disease Control and Prevention (CDC) Increasing prevalence of parent-reported
 524 attention-deficit/hyperactivity disorder among children—United States, 2003 and 2007. *MMWR*
 525 *Morb Mortal Wkly Rep*. 2010;59(44):1439–1443.
 526 13 Cussen A, Sciberras E, Ukoumunne OC, et al. Relationship between symptoms of attention-
 527 deficit/hyperactivity disorder and family functioning: a community-based study. *Eur J Pediatr*
 528 2012; 171: 271-280.
 529 14 Fischer, M. (1990). Parenting stress and the child with attention deficit hyperactivity disorder.
 530 *Journal of Clinical Child Psychology*, 19, 337–346.
 531 15 Patterson, G. R., DeBaryshe, B. D., Ramsey, E. (1989). A developmental perspective on anti-
 532 social behavior. *American Psychologist*, 44, 329–335.
 533 16 Biederman J, Faraone SV, Spencer TJ, et al. Functional impairments in adults with self-
 534 reports of diagnosed ADHD: A controlled study of 1001 adults in the community. *J Clin*
 535 *Psychiatry* 2006; 67: 524-540.
 536 17 Le HH, Hodgkins P, Postma MJ, et al. Economic impact of childhood/adolescent ADHD in a
 537 European setting: the Netherlands as a reference case. *Eur Child Adolesc Psychiatry* 2014; 23:
 538 587-598.
 539 18 Kim Y and Chang H. Correlation between attention deficit hyperactivity disorder and sugar
 540 consumption, quality of diet, and dietary behavior in school children. *Nutr Res Pract*. 2011 Jun;
 541 5(3): 236–245. doi: 10.4162/nrp.2011.5.3.236.
 542 19 Jo JI, Kim HK. Food habits and eating snack behaviors of middle school students in Ulsan
 543 area. *Korean J Nutr*. 2008;41:797–808.
 544 20 Harvard Medical School. Harvard Mental Health Letter: Diet and attention deficit
 545 hyperactivity disorder. [http://www.health.harvard.edu/newsletter_article/Diet-and-attention-](http://www.health.harvard.edu/newsletter_article/Diet-and-attention-deficit-hyperactivity-disorder)
 546 [deficit-hyperactivity-disorder](http://www.health.harvard.edu/newsletter_article/Diet-and-attention-deficit-hyperactivity-disorder)
 547 21 Silverman, B. L., Rizzo, T. A., Cho, N. H., Metzger, B. E. (1998) Long-term effects of the
 548 intrauterine environment. *Diabetes Care* 21,B142-B149.
 549 22 Plagemann, A., Harder, T., Kohlhoff, R., Rhode, W., Dorner, G. (1997) Overweight and
 550 obesity in infants of mothers with long-term insulin-dependent diabetes or gestational
 551 diabetes. *Int. J. Obes. Rel. Metabol. Disord*. 21,451-456.
 552 23 Muhlhausler BS, Adam CL, Findlay PA, Duffield JA, McMillen IC. Increased maternal
 553 nutrition alters development of the appetite-regulating network in the brain. *The FASEB Journal*
 554 2006; vol. 20(8):1257-1259. doi:10.1096/fj.05-5241fje
 555 <http://www.fasebj.org/content/20/8/1257.full>
 556 24 Ong ZY, Muhlhausler BS. Maternal "junk-food" feeding of rat dams alters food choices and
 557 development of the mesolimbic reward pathway in the offspring. *FASEB J*. 2011;
 558 Jul;25(7):2167-79. doi: 10.1096/fj.10-178392. Epub 2011 Mar 22.
 559 25 O'Neill, Brendon. (2006). "Is this what you call junk food?". BBC News.
 560 http://news.bbc.co.uk/2/hi/uk_news/magazine/6187234.stm.
 561 26 Centers for Disease Control and Prevention (CDC). Data & Statistics. Attention-Deficit /
 562 Hyperactivity Disorder (ADHD). New Data: Medication and Psychological Services Among
 563 Children Ages 2-5 Years (Healthcare Claims Data). <http://www.cdc.gov/ncbddd/adhd/data.html>

564 27 Johnson RJ, Gold MS, Johnson DR, Ishimoto T, Lanaspa MA, Zahniser NR, Avena NM.
 565 Attention-Deficit/Hyperactivity Disorder: Is it Time to Reappraise the Role of Sugar
 566 Consumption? *Postgrad Med.* 2011 Sep; 123(5): 39–49. doi: 10.3810/pgm.2011.09.2458.
 567 <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3598008/>
 568 28 Sullivan EL, Nousen L, Chamlou K. Maternal High Fat Diet Consumption during the
 569 Perinatal Period Programs Offspring Behavior *Physiol Behav.* 2014 Jan 17; 123:
 570 10.1016/j.physbeh.2012.07.014. doi: 10.1016/j.physbeh.2012.07.014.
 571 29 Swift, K. D., Sayal, K. and Hollis, C. (2014), ADHD and transitions to adult mental health
 572 services: a scoping review. *Child: Care, Health and Development*, 40: 775–786.
 573 doi: 10.1111/cch.12107.
 574 30 Stevenson, J., Buitelaar, J., Cortese, S., Ferrin, M., Konofal, E., Lecendreux, M., Simonoff,
 575 E., Wong, I. C. K., Sonuga-Barke, E. (2014), Research Review: The role of diet in the treatment
 576 of attention-deficit/hyperactivity disorder – an appraisal of the evidence on efficacy and
 577 recommendations on the design of future studies. *Journal of Child Psychology and Psychiatry*,
 578 55: 416–427. doi: 10.1111/jcpp.12215.
 579 31 Tarver, J., Daley, D. and Sayal, K. (2014), Attention-deficit hyperactivity disorder (ADHD):
 580 an updated review of the essential facts. *Child: Care, Health and Development*, 40: 762–774.
 581 doi: 10.1111/cch.12139.
 582 32 Ahmed, R., McCaffery, K. J. and Aslani, P. (2015), Development and validation of a question
 583 prompt list for parents of children with attention-deficit/hyperactivity disorder: a Delphi study.
 584 *Health Expectations*. doi: 10.1111/hex.12341.
 585 33 Nigg JT, Lewis K, Edinger T, et al. Meta-analysis of attention-deficit /
 586 hyperactivity disorder or attention-deficit/hyperactivity disorder symptoms, restriction diet, and
 587 synthetic food color additives. *J Am Acad Child Adolesc Psychiatry.* 2012; 51:86-97.
 588 34 Nikolas, M. A., Klump, K. L. and Burt, S. A. (2015), Parental Involvement Moderates
 589 Etiological Influences on Attention Deficit Hyperactivity Disorder Behaviors in Child Twins.
 590 *Child Development*, 86: 224–240. doi: 10.1111/cdev.12296.
 591 35 Paidipati, C. P. and Deatrick, J. A. (2015), The Role of Family Phenomena in Children and
 592 Adolescents With Attention Deficit Hyperactivity Disorder. *Journal of Child and Adolescent*
 593 *Psychiatric Nursing*, 28: 3–13. doi: 10.1111/jcap.12097
 594 36 Lee, S. I., Schachar, R. J., Chen, S. X., Ornstein, T. J., Charach, A., Barr, C. and Ickowicz, A.
 595 (2008), Predictive validity of DSM-IV and ICD-10 criteria for ADHD and hyperkinetic disorder.
 596 *Journal of Child Psychology and Psychiatry*, 49: 70–78. doi: 10.1111/j.1469-7610.2007.01784.
 597 37 Pringsheim, T. and Steeves, T. (2012), Cochrane Review: Pharmacological treatment for
 598 Attention Deficit Hyperactivity Disorder (ADHD) in children with comorbid tic disorders. *Evid.-*
 599 *Based Child Health*, 7: 1196–1230. doi: 10.1002/ebch.1861.
 600 38 Arnold LE, Lofthouse N, Hurt E. Artificial food colors and attention-deficit/hyperactivity
 601 symptoms: conclusions to dye for. *Neurotherapeutics.* 2012; 9:599-609.
 602 39 Stevenson J, Bateman B, Warner JO. Rejoinder to Eigenmann PA, Haengelli CA, Food
 603 colourings and preservatives–allergy and hyperactivity (*Lancet* 2004; 364:823–4) and an
 604 erratum. *Arch Dis Child.* 2005;90(8):875.
 605 40 Johnson, Paul M.; Kenny, Paul J. (2010). "Addiction-like reward dysfunction and compulsive
 606 eating in obese rats: Role for dopamine D2 receptors". *Nature Neuroscience* 13 (5): 635–41.
 607 doi:10.1038/nn.2519. PMC 2947358. PMID 20348917.

41 Ong ZY and Muhlhausler BS. Maternal "Junk-food" feeding of rat dams alters food choices and development of the mesolimbic reward pathway in the offspring, *FASEB J* Jul 2011; 25: 2167-2179).

42 Wright, N., Moldavsky, M., Schneider, J., Chakrabarti, I., Coates, J., Daley, D., Kochhar, P., Mills, J., Sorour, W. and Sayal, K. (2015), Practitioner Review: Pathways to care for ADHD – a systematic review of barriers and facilitators. *Journal of Child Psychology and Psychiatry*, 56: 598–617. doi: 10.1111/jcpp.12398.

43 Young, J. T., Carruthers, S., Kaye, S., Allsop, S., Gilsenan, J., Degenhardt, L., van de Glind, G., van den Brink, W. and Preen, D. (2015), Comorbid attention deficit hyperactivity disorder and substance use disorder complexity and chronicity in treatment-seeking adults. *Drug and Alcohol Review*, 34: 683–693. doi: 10.1111/dar.12249.

44 Eaves LJ, Silberg JL, Meyer JM, Maes HH, Simonoff E, Pickles A, et al. Genetics and Developmental Psychopathology: 2. The Main Effects of Genes and Environment on Behavioral Problems in the Virginia Twin Study of Adolescent Behavioral Development. *Journal of Child Psychology and Psychiatry* Vol. 38(8): 965–980, November 1997. DOI: 10.1111/j.1469-7610.1997.tb01614.x <http://onlinelibrary.wiley.com/doi/10.1111/j.1469-7610.1997.tb01614.x/full>.

45 Bergen SE, Gardner CO, Kendler KS. Age-Related Changes in Heritability of Behavioral Phenotypes over Adolescence and Young Adulthood: A Meta-Analysis. *Twin Research and Human Genetics* Vol.10(3);2007: 423-433. <http://journals.cambridge.org/action/displayAbstract?fromPage=online&aid=8492271&fileId=S1832427400007994>.

46 Wiles NJ, Northstone K, Emmett P, Lewis G. 'Junk food' diet and childhood behavioural problems: results from the ALSPAC cohort. *European Journal of Clinical Nutrition* (2009) 63, 491–498; doi:10.1038/sj.ejcn.1602967. <http://www.nature.com/ejcn/journal/v63/n4/abs/1602967a.html>

47 Kim Y, Chang H. Correlation between attention deficit hyperactivity disorder and sugar consumption, quality of diet, and dietary behavior in school children. *Nutr Res Pract*. 2011 Jun;5(3):236-245. <http://dx.doi.org/10.4162/nrp.2011.5.3.236>

48 Sagvolden T. Behavioral validation of the spontaneously hypertensive rat (SHR) as an animal model of attention-deficit/hyperactivity disorder (AD/HD). *Neuroscience & Biobehavioral Reviews* Vol. 24(1); 2000: 31–39. <http://www.sciencedirect.com/science/article/pii/S0149763499000585>

49 katya rubia (2005). ADHD under the “micro-scope” of the rat model. *Behavioral and Brain Sciences*, 28, pp 439-440 doi:10.1017/S0140525X05350072). <http://journals.cambridge.org/action/displayAbstract;jsessionid=68218EBF43483C776117BC47F5B4AFE2.journals?fromPage=online&aid=332983>

50 American Psychiatric Association: Diagnostic and Statistical Manual of Mental Diseases (DSM-IV), 4th ed. Washington, DC, American Psychiatric Publishing, 1994.

51 Dobie C (2012). "Diagnosis and management of attention deficit hyperactivity disorder in primary care for school-age children and adolescents". Institute for Clinical Systems Improvement: 79. https://www.icsi.org/_asset/60nzc5/ADHD-Interactive0312.pdf

52 Centers for Disease Control and Prevention (CDC). National Center on Birth Defects and Developmental Disabilities. "Facts about ADHD". <http://www.cdc.gov/ncbddd/adhd/facts.html>.

53 Valdizán, JR; Izaguerri-Gracia, AC (2009). "Attention deficit hyperactivity disorder in adults". *Revista de neurologia* 48 Suppl 2: S95–9.

54 American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Arlington: American Psychiatric Publishing. pp. 59–65. ISBN 0890425558.

55 National Institute of Mental Health (2008). "Attention Deficit Hyperactivity Disorder (ADHD)". National Institutes of Health. <http://www.nimh.nih.gov/health/topics/attention-deficit-hyperactivity-disorder-adhd/>

56 Bailey Eileen. "ADHD and Learning Disabilities: How can you help your child cope with ADHD and subsequent Learning Difficulties? There is a way." Remedy Health Media, LLC.

57 Singer HS. "Tourette syndrome and other tic disorders". *Handb Clin Neurol*. 2011;100:641–57. doi:10.1016/B978-0-444-52014-2.00046-X.

58 McBurnett K, Pfiffner LJ. (2009). "Treatment of aggressive ADHD in children and adolescents: Conceptualization and treatment of comorbid behavior disorders". *Postgrad Med* 121(6): 158–165. doi:10.3810/pgm.2009.11.2084

59 OLD Biederman J, Faraone S, Wozniak EM, Chen L, Ouellette C, Marris A, et al. Attention-Deficit Hyperactivity Disorder and Juvenile Mania: An Overlooked Comorbidity? *Adolescent Psychiatry* Vol. 35(8); 1996: 997–1008. <http://www.sciencedirect.com/science/article/pii/S089085670962493X> and substance abuse.

60 Lee SS, Humphreys KL, Flory K, Liu R, Glass K. Prospective association of childhood attention-deficit/hyperactivity disorder (ADHD) and substance use and abuse/dependence: A meta-analytic review. *Clinical Psychology Review* Vol. 31(3); 2011: 328–341. <http://www.sciencedirect.com/science/article/pii/S0272735811000110>

61 OLdTArver J., Daley D. and Sayal K. (2015). Beyond symptom control for attention-deficit hyperactivity disorder (ADHD): what can parents do to improve outcomes? *Child: Care, Health and Development*, 41: 1–14. doi: 10.1111/cch.12159

62 Thapar A, Cooper M, Eyre O, Langley K. (2013), Practitioner Review: What have we learnt about the causes of ADHD? *Journal of Child Psychology and Psychiatry*, 54: 3–16. doi: 10.1111/j.1469-7610.2012.02611.

63 Pelham, W. E., Lang, A. R., Atkeson, B., Murphy, D. A., Gnagy, E. M., Greiner, A. R., et al. (1998). Effects of deviant child behavior on parental alcohol consumption: Stress-induced drinking in parents of ADHD children. *American Journal of Addictions*, 7, 103–114.

64 Patterson, G. R., & Guillon, M. E. (1968). *Living with children: New methods for parents and teachers*. Champaign, IL: Research Press.

65 Forehand, R. L., & McMahon, R. M. (1981). *Helping the noncompliant child: A clinician's guide to parent training*. New York: Guilford.

66 Chronis AM, Chacko A, Fabiano GA, Wymbs BT, and Pelham WE, Jr. Enhancements to the Behavioral Parent Training Paradigm for Families of Children With ADHD: Review and Future Directions. *Clinical Child and Family Psychology Review*, Vol. 7, No. 1, 2004; p. 1-27.

67 Horn, W. F., Ialongo, N., Greenberg, G., Packard, T., & SmithWinberry, C. (1990). Additive effects of behavioral parent training and self-control therapy with attention deficit hyperactivity disordered children. *Journal of Clinical Child Psychology*, 19, 98–110; “

68 MTA Cooperative Group. (1999a). 14-month randomized clinical trial of treatment strategies for attention deficit hyperactivity disorder. *Archives of General Psychiatry*, 56, 1073–1086.

69 Barkley, R. A., Shelton, T. L., Crosswait, C., Moorehouse, M., Fletcher, K., Barrett, S., et al. (2000). Multi-method psychoeducational intervention for preschool children with disruptive behavior: Preliminary results at posttreatment. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 41, 319–332.

698 70 Webster-Stratton, C., Reid, M. J., & Hammond, M. (2001a). Preventing conduct problems,
699 promoting social competence: A parent and teacher training partnership in head start. *Journal of*
700 *Clinical Child Psychology*, 30, 283–302.

701 71 Rowland, A. S., Umbach, D. M., Stallone, L., Naftel, J., Bohlig, E. M., & Sandler, D. P.
702 (2002). Prevalence of medication treatment for attention deficit-hyperactivity disorder among
703 elementary school children in Johnston County, North Carolina. *American Journal of Public*
704 *Health*, 92, 231–234.

705 72 Pelham, W. E. (1999). The NIMH multimodal treatment study for attention-deficit
706 hyperactivity disorder: Just say yes to drugs alone? *Canadian Journal of Psychiatry*, 44, 981–990.

707 73 Biederman J, Faraone S, Wozniak EM, Chen L, Ouellette C, Marris A, et al. Attention-Deficit
708 Hyperactivity Disorder and Juvenile Mania: An Overlooked Comorbidity? *Adolescent*
709 *Psychiatry* Vol. 35(8); 1996: 997–1008.

710 74 Magee E. Diet & weight management. Junk-food facts. 2016. Webmed.
711 <http://www.webmd.com/diet/junk-food-facts>

712 75 Centers for Disease Control and Prevention (CDC). Data & Statistics. Attention-Deficit /
713 Hyperactivity Disorder (ADHD). New Data: Medication and Psychological Services Among
714 Children Ages 2-5 Years (Healthcare Claims Data). <http://www.cdc.gov/ncbddd/adhd/data.html>